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URANIUM POISONING

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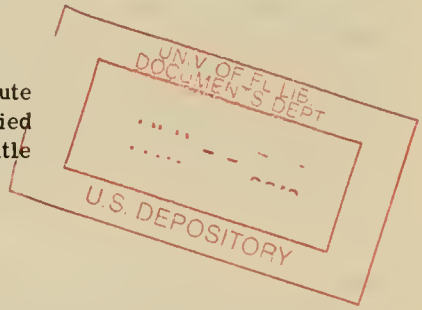

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URANIUM POISONING

By Sol Wexler

ABSTRACT

Poisoning from uranium may come about either through inhalation of uranium oxide dust (U_3O_8, UO_2, or UO_3) or through contact with the soluble salts.

The pathological manifestation of uranium poisoning is nephritis, an inflammation of the kidney. The intensity of the anatomical and functional lesion is the result of the accumulation of the metal in the organ as a result of attempts at excretion.

Protection of the body against uranium involves protection of the hands and care to avoid inhalation or ingestion. In the synthesis of uranyl nitrate, rubber gloves are recommended. Protection against ingestion depends solely upon the worker; care must be exercised to prevent the taking of any uranium by mouth during such operations as pipetting.

* * * * *

For more than one hundred years, uranium has been known to be poisonous to the animal organism. As it is as toxic as arsenic, it is included in the official lists of poisons in Germany and Russia. In particular, uranosic oxide, U_3O_8, is liberated as a dust in industries in which uranium is exposed to high temperatures and, without protection, may be inhaled and swallowed by workers. Since U_3O_8 is soluble in gastric juice, ingestion of the oxide either directly or following inhalation would probably lead to manifestations of poisoning. The symptoms are similar to those of other heavy metal poisonings, expecially those due to certain organic arsenic compounds. The poisonous effect of uranium is usually ascribed to the action of the metal as such. Finally, uranium is not a rapid poison, and the action of small and large doses is the same.

The first noticeable symptom of uranium poisoning is muscular weakness. Although no case of glucose in the urine was observed, in a study of a group of four workers in a Belgian uranium salt preparation factory, glycosuria is a symptom of uranium poisoning and is due to the lowered glucose threshold of the kidneys. Furthermore, uranium salts, especially the nitrate, have an inhibitory effect on the activity of the digestive ferments, particularly fptylin and trypsin. In addition, the salts check digestion, but apparently increase protein metabolism to a slight extent. Uranium increases the output of carbon dioxide, raises the body temperature, and finally leads to emaciation. Being an irritant poison, it produces gastro-intestinal disorder of greater or less intensity. Concentrated solutions of uranium salts corrode the mucous membrane transforming the walls of the stomach into a dead film of uranic albuminate.

The greatest pathological effect of uranium poisoning is nephritis, an inflammation of the kidney. It is a diffuse, degenerative lesion affecting the renal parenchyma, the interstitial tissue, and the renal vascular system. There are a number of types of nephritis depending on the part of the kidneys attacked. Acute nephritis is the destruction of the renal tissue, similar to that produced by arsenic, mercury, and phosphorus. Glomerular nephritis refers to inflammation of the glomeruli of the kidneys. In interstitial and chronic nephritis, such as that due to lead poisoning and alcohol, the kidneys become small, cystic, nodulated, and adherent to their capsules; furthermore, the interstitial tissue is increased and there is a thickening of the vessel walls and the malpighian corpuscles.

In addition, the heart becomes hypertrophied, and the walls of the small arteries thicken. Nervous symptoms, such as loss of sight and coordinating power, sometimes predominate as a result of the coagulating action of uranium salts on albumin. Albumin in the urine (albuminuria) is the earliest indication of renal irritation, which functionally at least is the earliest demonstrable stage of nephritis.

Whereas uranium as a poison produces widespread lesions from the functional point of view, there is no doubt that anatomically its most marked effect is on the kidney. Experiments show a marked tendency for the deposition of uranium in the kidneys, with no detectable amounts in any of the other organs; the deposition is exclusively in the renal cortex and not in the medullary portion of the kidneys. The fact that this deposition occurs in living animals and not in excised kidneys would indicate that it has to do with living processes. This raises the question as to whether it is due to a special lack of resistance of the kidney to the poison, a special affinity for the poison, or the attempted excretion through the kidneys. It appears, as a result of experimental uranium nephritis, that the intensity of the anatomical and functional lesion is not to be attributed to a susceptibility of this organ for the poison, but is the result of the accumulation of the metal in the organ as a result of attempts at excretion. This would result in a vicious circle in which increased attempts at excretion result in increased concentration of the poison because of decreased effectiveness of excretion.

In the study of four Belgian workers, two of whom worked with soluble and two with insoluble uranium salts, it was shown that there was a decrease in the nitrogen and a large increase in the chlorine content of the urine. Furthermore, a small amount of uranium was found in the urine. A small dose of uranium increases the volume of urine. However, it is well known that in uranium poisoning there is, in general, a considerable reduction in the urine output, which may go on to complete suppression, this process going hand in hand with degenerative processes in the renal epithelium and later alterations in the glomerulus. This can be explained at least in part by the early collection of uranium in the kidney, the uranium operating by its coagulative effect on the epithelial protoplasm producing haemorrhages in the kidney.

Accompanying nephritis is an acid intoxication which may be reduced by the administration of sodium bicarbonate. Also concurrent are marked changes in the parathyroid and thyroid glands.

In the study of the four Belgian uranium workers it has been found that the uranium salts under consideration (sodium uranate and uranyl nitrate) clearly have an effect upon the blood forming processes, resulting in every case in a considerable decrease in the leucocyte content. This was particularly marked in the two workmen handling insoluble salts, their skin and mucous membrane having come in contact with large quantities of uranium. The blood of these workers also showed a decrease in the hematin content, with signs of anemia, those who handled the soluble salts being the more affected in this way. A worker who handled the salts six months exhibited purpling of the hands, this being the result of alteration in the red blood corpuscles. This pale color showed anemia, the confirmation of which was given by the results of the blood and urine analysis. Examination showed that the red blood cell counts for the four workers were not equal. Some cells of the soluble salt workers were deformed; in the case of one of the workers there was a deformation of the red blood cell nuclei. On the other hand, the corpuscles of the insoluble salt workers were normal.

Lastly, it must be mentioned that the salts deposited on the skin and mucous membrane are also radioactive poisoning agents. The pathological effects and methods of protection have been described in the report "Radium: Pathological Effects and Protection." However, because of the very long half-life of uranium (10^6 years), its rate of bombardment is not very large, and, consequently, the danger from this source is much less than in the case of radium.

Protection of the body against uranium involves protection of the hands and protection against ingestion and inhalation, expecially of U_3O_8. In the synthesis of uranyl nitrate, rubber gloves are

recommended. Protection against ingestion depends solely upon the worker; care must be taken to prevent the taking of any uranium by mouth during such operations as pipetting. It is recommended that masks with breathing screens be worn during boiling or grinding of uranium salts, it being advisable that the mask cover the entire face of the worker. It is practically impossible to have no contact with uranium. However, it has been shown that absorption of uranium salts by the skin is extremely small.

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